vant history data included HBP, chronic virus C hepatitis, and allergy to quinolones.

In 2007, the patient attended the emergency room complaining of fever, dry cough, and some abdominal discomfort.

On physical examination, the patient was found to be in a good general condition, with a baseline SatO of 94%, BP values of 130/60 mmHg, and a temperature higher than 38 ºC. The only remarkable findings in cardiopulmonary auscultation were crackling sounds, particularly in the left base. Cysts of her underlying disease were palpated in the abdomen.

Results of blood laboratory test son admission included: Creatinine 1.5 mg/dL, WBC 5090 (82% neutrophils), platelets 95,000, prothrombin activity 55%. Results of all other blood tests and urine analysis were normal.

Chest X-rays showed blurring of left base, and patient was admitted to hospital with a diagnosis of left basal pneumonia.

After collecting samples for culture (blood, urine, sputum), empirical treatment was started with cefotaxime.

At 24 hours of admission, the patient experienced impairment of her general condition, severe arterial hypotension, oliguria, and coagulopathy, requiring hemodynamic support with dopamine and colloids, furosemide infusion, and vitamin K treatment. A CT scan of the abdomen and pelvis found no added disease to cysts.

Because of severity of the condition, cefotaxime was replaced by imipenem and clarythromycin. Blood culture results reported isolation of penicillin-resistant and imipenem-susceptible *Hafnia alvei.*

Measures taken achieved clinical stability and improvement of the patient.

Immunosuppression, necessary to prevent rejection of transplanted organs, implies a predisposition to experience severe infection.[1] Rubin et al reported a calendar of infections depending on the transplant period: hospital-acquired infections are common in the first month, opportunistic infections predominate between the first and sixth months, and any community-acquired infection may occur after 6 months.[2]

A community-acquired *H. alvei* infection occurring in a kidney transplant patient 20 years after transplant is reported here.

Members of the *Hafnia* genus are Gram-negative bacilli belonging to the family Enterobacteriaceae that occasionally cause infection in humans. Though this species has been known since 1954, for decades it was considered to belong to the genus *Enterobacter*, and was therefore called «*Enterobacter hafniae*» or «*Enterobacter alvei*». Recent DNA and biochemical studies defined these organisms as members of a separate genus with a single species, *Hafnia alvei*.[3]

The gastrointestinal tract of humans and animals, particularly mammalians, but also birds, may be a reservoir for *Hafnia.*[3] Epidemics of *H. alvei* infection related to the poultry industry are well documented in the literature.[4] In our case, the patient was questioned again and said that she was in continuous contact with poultry, but had no awareness that her animals were ill.

Most patients colonized or infected by *H. alvei* have underlying diseases such as cancer (particularly hematological tumors), surgery, trauma, pulmonary disease, cirrhosis, hepatitis, or pancreatitis.[5] In the case reported, CRF for polycystic liver and kidney disease, chronic virus C hepatitis, and immunosuppression also existed. In this patient, comorbid conditions and repeated contact with poultry may have promoted infection development.

Very little is known about this microorganism as a pathogen in animals and humans. It may cause a great variety of systemic infections, including septicemia and pneumonia.

A literature review revealed that half the patients with bacteremia were immunocompromised because of tumors, hepatic disease, or HIV infection.[3] In transplant patients, association was only found with liver[6] and stem cell transplant.[7] Our case is the first reported in a kidney transplant patient.

In conclusion, immunosuppression associated to transplant and underlying diseases predispose to uncommon infections. A first description of an *H. alvei* infection in a kidney transplant patient whose main clinical manifestation was septicemia is given.

1. Garibaldi RA. Infections in organ transplant recipient. *Infect Control* 1983; 4 (6): 460-464.
2. Rubin RH, Wolfson JS, Cosimi AB, Tolkoff-Rubin NE. Infection in the renal transplant recipient. *Am J Med* 1981; 70 (2): 405-411.
3. Janda JM, Abbott SL. The genus Hafnia: from soup to nuts. *Clin Microbiol Rev* 2006; 19 (1): 12-18.
4. Real F, Fernández A, Acosta F, Acosta B, Castro P, Déniz S, Orós J. Septicemia associated with Hafnia alvei in laying hens. *Avian Dis* 1997; 41: 741-747.
5. Günthard H, Pennekamp A. Clinical significance of extraintestinal Hafnia alvei isolates from 61 patients and review of the literature. *Clin Infect Dis* 1996; 22: 1040-1045.
6. Barry JW, Domínguez EA, Boken DJ, Preheim LC. Hafnia alvei infection after liver transplantation. *Clin Infect Dis* 1997; 24 (6): 1263-1264.
7. Candoni A, Trevisan R, Fili C, Tiribelli M, Fanin R. Abdominal abscess and Hafnia alvei septicemia ocurring during the aplastic phase after autologous stem-cell transplantation in a patient with diffuse large B-cell lymphoma. *J Infect Chemother* 2004; 10 (5): 303-306.

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Segmental renal artery stenosis causing renovascular hypertension

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To the editor: Renovascular hypertension (RVH) is the most common form of secondary hypertension, with a prevalence ranging from 5%-15%.[1] RVH is caused by stenosis of the renal artery of its branches that induces renal hypoperfusion leading to activation of the renin-angiotensin system and subsequent occurrence of hypertension.[2] Atherosclerotic

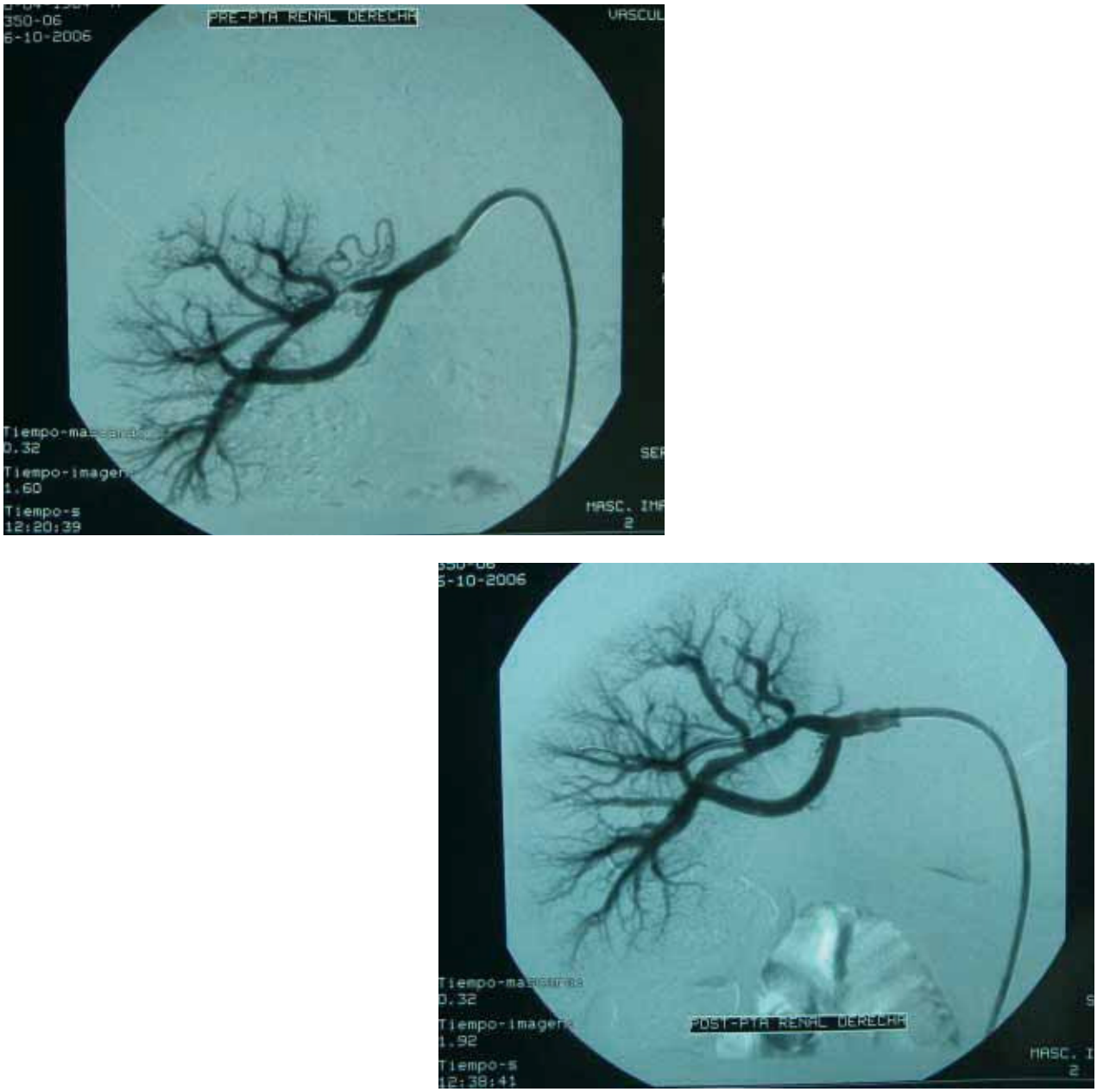

Figure 1.

disease is the most common cause of RVH, and lesions usually occur in the first few centimeters of the renal artery and may involve a large area. Fibromuscular disease is the most common cause of RVH in young women, and diagnosis should be considered in people with early onset of severe hypertension, though it is sometimes and incidental finding. It is important that RVH is recognized, because it responds well to surgical treatment. These lesions often arise beyond the first few centimeters of the renal artery and may be associated to disease in more distal branches.

We report the case of a 22-year-old female patient who attended the department of nephrology for arterial hypertension. She had no remarkable personal or family history. Physical examination revealed a sitting blood pressure of 175/100 mmHg and a heart rate of 76 beats per minute. Examination was otherwise unremarkable. Supplemental tests, including complete blood count, biochemistry, urinary catecholamines, urine sediment, chest X-rays, and electrocardiogram, were normal. Treatment was started with bisoprolol, and an angio-MRI was requested for renal study. At 3 months of treatment, the patient showed a slight improvement in her blood pressure values (160/90 mmHg), and angio-MRI revealed doubtful fibrodysplasia at left renal artery level. Renal arteriography was therefore performed for confirmation. Arteriography showed critical stenosis in the right renal segmental artery

with collateral circulation and no significant lesions in left renal artery. Angioplasty was performed on this stenosis using a 4 x 20 mm balloon catheter with good morphological result and disappearance of collateral circulation (fig. 1). At 2 years of follow-up, the patient has normal blood pressure values under no drug treatment.

Fibromuscular dysplasia usually occurs in young women aged 15-50 years, and the arterial sector usually affected are the distal 2/3 of the renal artery. RVH caused by involvement of renal artery branches is rare, and in the reported cases the polar arteries[3] or the accessory arteries[4] were affected. In our case, RVH was caused by involvement of a main branch of the right renal artery. RVH resulting from involvement of this segmental artery is an exceptional case. As regards treatment, advances in endovascular procedures in recent years have allowed for a high success rate in therapeutic management of these patients.

1. J.Menard JP. Grunfeld. Hipertension Renovascular Tomo I 1982:189-202.
2. Strandness DE. Natural history of renal artey stenosis. *Am J Kidney Dis* 1994; 24: 630-635.
3. García-Gómez MC, Fernández-Fresnedo G, Sanz de Castro S, Arias M. Hipertensión arterial renovascular secundaria a estenosis de la arteria polar. *Nefrología* 2002; Vol XXII, n.º 1: 85-86.
4. Cuxart M, Picazo M, Matas M, Canalias J, Nadal C, Falcó J. Hipertensión arterial y estenosis de la arteria renal accesoria. *Nefrología* 2007; Vol XXVII n.º 4: 509-510.

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